DECEMBER 1961

Disease-a-Month

Tachycardias: Diagnosis and Treatment

W. PROCTOR HARVEY

YEAR BOOK MEDICAL PUBLISHERS • INC.
CHICAGO

DM

Disease-a-Month

MONTHLY CLINICAL MONOGRAPHS ON CURRENT MEDICAL PROBLEMS

RECENT AND FORTHCOMING ISSUES

Wallace N. Jensen—THE HEMOGLOBINOPATHIES

Robert J. Ryan—MALE HYPOGONADISM

Sol Katz, Joseph W. Peabody, Jr., and *Edgar W. Davis*—THE SOLITARY PULMONARY NODULE

Edward A. Gaensler—DYSPNEA: DIAGNOSTIC AND THERAPEUTIC IMPLICATIONS

Robert Ward and *Saul Krugman*—VIRAL HEPATITIS

Ronald S. Mintz—DETECTION AND MANAGEMENT OF THE SUICIDAL PATIENT

E. Charles Kunkle—COMA

Sherman M. Mellinkoff—ULCERATIVE COLITIS

Herbert S. Heineman and *Abraham I. Braude*—SHOCK IN INFECTIOUS DISEASES

Jackson A. Smith—PSYCHOACTIVE DRUGS

PRINTED IN U.S.A.

Tachycardias: Diagnosis and Treatment

W. PROCTOR HARVEY

TABLE OF CONTENTS

W. Proctor Harvey

is Professor of Medicine and Chief, Division of Cardiology, Georgetown University Medical Center. He received his M.D. degree from Duke University School of Medicine and took his intern and residency training at Peter Bent Brigham Hospital in Boston. Dr. Harvey is co-author, with Dr. Samuel A. Levine, of the textbook *Clinical Auscultation of the Heart*.

TACHYCARDIAS have assumed no less importance with the passage of time and with advances in medicine. It is apparent that, like the aging of man, the problems of the diagnosis and treatment of rapid heart action will always be with us. Most practicing physicians in their office or hospital practice have some patients with tachycardia, either benign or of grave significance. The aim of this discussion will be to outline a practical clinical approach to the recognition, treatment and prevention of the more common tachycardias: sinus tachycardia, paroxysmal atrial tachycardia, paroxysmal atrial fibrillation, atrial flutter, atrial tachycardia with block, nodal tachycardia, ventricular tachycardia and ventricular fibrillation. Without question, the electrocardiogram is the most important device for accurate diagnosis of the tachycardias, but at times this is not immediately available and the physician must use all possible clinical clues to aid in proper diagnosis and treatment. The necessity of paying detailed attention to clinical features (including the rate, rhythm, first heart sound and the effect of carotid sinus stimulation) as well as the electrocardiographic aspects will be stressed.

NORMAL SINUS TACHYCARDIA

Rate

A heart rate of over 100 beats per minute is designated sinus tachycardia. The beat originates in normal fashion in the sino-atrial node and travels down the usual conduction pathways to

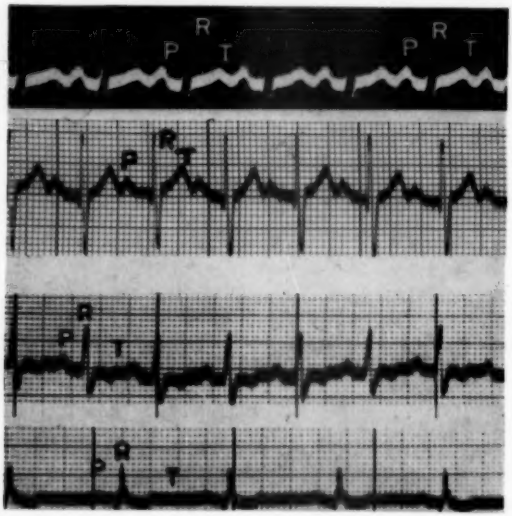

Fig. 1.—Three patients with sinus tachycardia. In the upper tracing, the tachycardia is associated with lupus erythematosus (rate 116). The middle tracing is of a girl, aged 7, with atrial septal defect and advanced congestive heart failure. Lower two tracings are from man, 47, with congestive heart failure. Sinus tachycardia of 120 slowed to rate of 78 (*bottom tracing*, with different lead on ECG) following therapy.

the ventricles, differing from the normal sinus rhythm only in that the rate is faster. As a rule the accelerated heart rate varies between 100 and 150 per minute and may be noted with exercise, emotional episodes, fever, infections, hyperthyroidism,

beriberi heart disease, anemia and in some patients with cor pulmonale. Active carditis from rheumatic fever or other forms of myocarditis may also result in sinus tachycardia. In some persons the increase in rate follows smoking or the ingestion of alcoholic beverages or coffee. In others the tachycardia may represent a vagal release after taking drugs such as atropine or those that have an atropine-like action; epinephrine and ephedrine also result in a faster rate than normal. Sinus tachycardia is seen concomitant with shock states, significant hypotension and/or acute blood loss. A more rapid heart rate is normal in infants and children, generally averaging 120 a minute and slowing to a normal range in the early teens. If an adult has a heart rate over 150, it is probable that a mechanism other than the normal sinus is present, but on occasion such a rate may represent sinus tachycardia. In infants and children sinus tachycardia at times reaches a rate of 180–200. It is helpful to know the patient's normal heart rate so that the actual increase may be used in evaluating the cause of the sinus tachycardia. An elevated rate may be a compensatory adjustment in congestive heart failure, particularly when noted in conjunction with a ventricular diastolic gallop, either or both representing early or subtle findings of underlying heart disease and congestive heart failure. This is well illustrated in Figure 1. The top electrocardiogram is that of a woman with the myocarditis of lupus erythematosus. The second tracing is that of a child with atrial septal defect and congestive failure, and the other two tracings are those of a patient with hypertensive heart disease and congestive heart failure. A ventricular gallop was heard in all of these patients.

Rhythm

The rhythm in normal sinus tachycardia is essentially regular, although some variation in rate may be observed over a period of minutes or hours (e.g., the rhythm that is regular at a rate of 130 remains so though the rate may be 116 per minute 20–40 minutes later). This is in contradistinction to paroxysmal atrial

tachycardia in which the same rate is likely to be observed throughout.

First Heart Sound

This remains constant in intensity except for the minor variations that occur normally with changes in respiration. The first heart sound with sinus tachycardia, as in other tachycardias, is generally accentuated (Fig. 2).

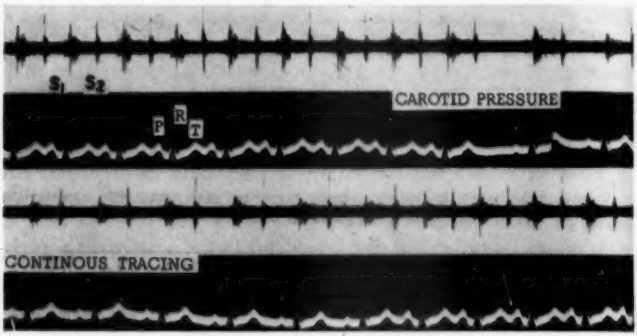

Fig. 2.—Sinus tachycardia with rate of 110 slows with carotid sinus pressure and gradually returns to original rate. Note constant first sound (S_1) before and after carotid pressure.

Carotid Sinus Stimulation

A typical response to carotid sinus pressure (see Figures 2 and 6) is a gradual slowing of the heart rate with a gradual return to its original rate. Many patients are unaware that they have an elevated heart rate. Others complain of palpitation which may appear to the patient as a faster rate than is actually present, particularly if this is of a forceful or "pounding" type. When such a patient is examined during periods of no com-

plaint of palpitation, a careful history is often rewarding in that the patient may be able to describe the symptoms after the physician has illustrated by movement of his hand placed over his own precordium to simulate the various types of regular (or irregular) rapid heart actions, varying the rate from slow to quite rapid (Fig. 3).

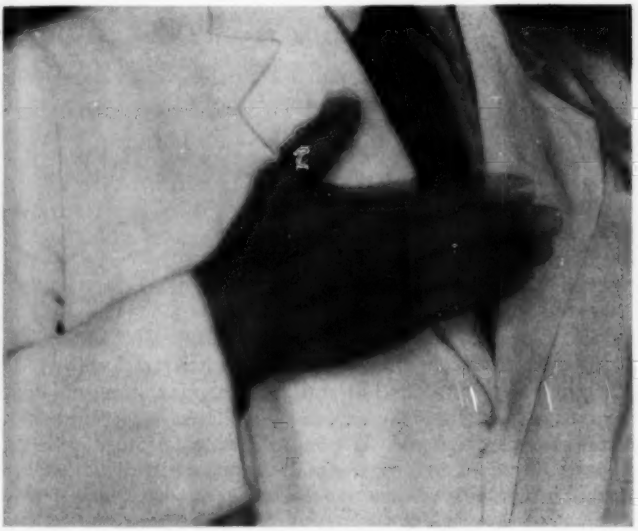

FIG. 3.—Physician demonstrating simulated type of rapid heart action. The patient can often give valuable clues to his own arrhythmia after this simple demonstration. (Physician's hand photographed while in motion.)

ELECTROCARDIOGRAM

The normal P wave can generally be seen as in Figures 1 and 2; with faster rates having shortened diastoles, the P wave may merge with the preceding T wave.

Treatment

The underlying precipitating condition is treated. If the patient exhibits any concern over his faster rate, reassurrance can be given. The use of drugs such as digitalis and quinidine are not indicated in this condition unless required for treatment of the basic heart disease (e.g., digitalis for congestive heart failure).

PAROXYSMAL ATRIAL TACHYCARDIA

Paroxysmal atrial tachycardia is a rhythm which arises suddenly in an ectopic focus in the atrium and is, in effect, a rapid series of atrial extrasystoles coming from this focus. In some cases it may represent a circus movement mechanism. The abrupt onset of a rapid regular rhythm may be transient, lasting only a few seconds or minutes, or it may persist for hours, days and in some cases even longer.

Rate

The sudden onset of a rapid regular heart beat with a rate of 180–190 is characteristic of paroxysmal atrial tachycardia. It generally varies between 150 and 250. In infants and children the rate is more likely to assume higher levels—even over 300. Atrial tachycardia is seen in persons having no other evidence of heart disease more commonly than in those with underlying organic heart disease. Some patients have only one attack; others have infrequent attacks at intervals of several years still others have distressing recurrences varying from every few hours to several weeks. It appears to be more prevalent in the nervous, tense person, who can generally give a good description of his tachycardia. Its sudden onset is seemingly unrelated to any particular event, although in some people it may be tied up with an emotional upset, sudden physical exertion or a certain motion of the body; palpitation is usually noted. The patient can often identify this from the physician's imitation as he moves his hands over his precordium (Fig. 3). A rapid regular rhythm is often

described as "fluttering" or "pounding." Symptoms include nervousness, apprehension, weakness, dizziness and, rarely, syncope. It is of interest that some patients are unaware that tachycardia has been present. As a rule, the patient with an otherwise healthy heart tolerates this paroxysm of rapid heart action without any difficulty, and in fact many carry on work or other activities. After cessation of the attack, which is also usually abrupt in offset, generally no residual myocardial damage or impairment is evident, either by electrocardiogram or by clinical evaluation. Occasionally, however, nonspecific S-T and T wave changes may

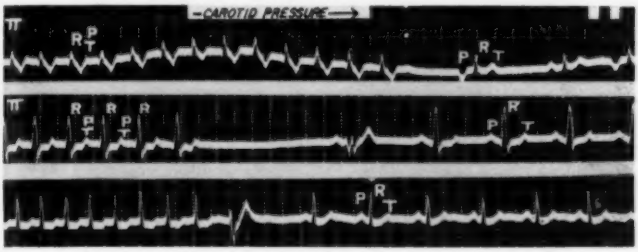

FIG. 4.—Three tracings of patients with atrial tachycardia reverting to normal sinus rhythm as a result of vagal stimulation. Note that P waves are difficult to identify with certainty when tachycardia is present.

be noted on the electrocardiogram, which persist for hours or days. These are designated as post-tachycardia changes and, in the absence of other evidence, may have no special significance. As illustrated in Figures 4 and 5, with a regular rapid rate the P wave is difficult to identify with certainty, and instead one sees the *R*-T, *R*-T complexes. At times, however, it is possible to identify the P wave. Response to carotid sinus pressure is shown where reversion to normal sinus rhythm takes place.

In patients with organic heart disease, the paroxysm of atrial tachycardia may be attended by significant symptoms, as is also true of any other type of rapid heart action. The patient with known rheumatic, hypertensive, arteriosclerotic or other heart disease, may be severely incapacitated by uncontrolled tachy-

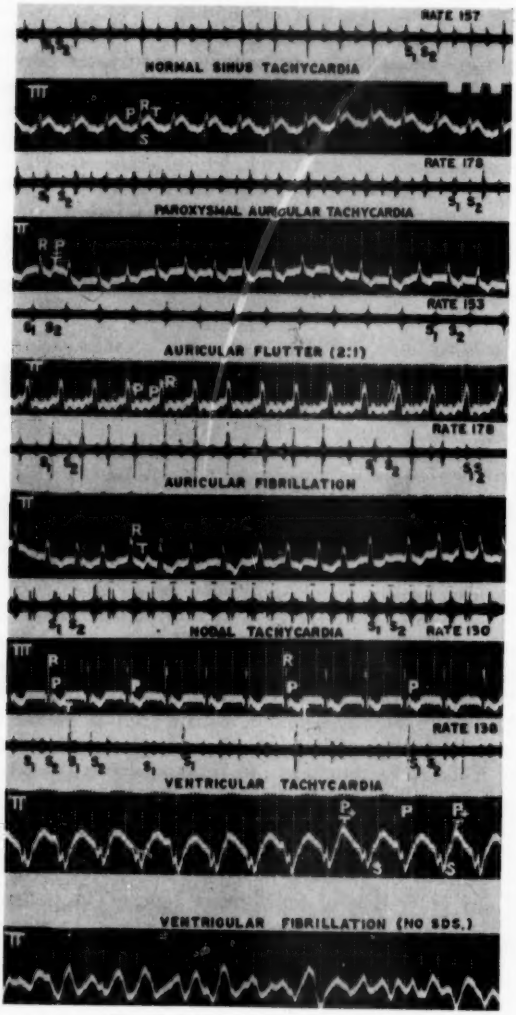

FIG. 5.—Legend on facing page.

cardia, particularly if it lasts for hours or days rather than minutes. Cardiac decompensation, angina, coronary insufficiency or even myocardial infarction may occur in a susceptible diseased heart. In some instances the blood pressure drops, and the pulse pressure becomes quite narrow, thereby predisposing to thrombosis of a peripheral vessel. Patients with coronary artery disease may also note severe precordial distress coincident with a rapid rate. Sometimes the type of pain characteristic of coronary insufficiency may be first observed during such a paroxysm. Patients with severe aortic insufficiency find that episodes of paroxysmal atrial tachycardia generally go badly with them. Precordial chest pain is frequent, and the abnormally forceful beat of the heart resulting from the severe leak of the aortic valve produces an even more forceful accentuation of the peripheral arterial signs of aortic insufficiency, thereby causing great annoyance as well as possible discomfort and/or cardiac decompensation. Occasionally a patient with paroxysmal atrial tachycardia presents a history of a precipitating event such as an emotional upset, undue nervousness, fatigue, indigestion, alcohol, particularly in excess, or in combination with a gallbladder attack.

If possible, the physician should observe the patient during the episode of paroxysmal atrial tachycardia. In this way he can arrive at an accurate diagnosis—making sure there is no confusion with other tachycardias. The heart rate during the attack remains constant in each patient; for example, a heart rate of 190 obtained after at least a full minute of careful counting will remain within a beat or two of this rate if checked an hour later, provided the tachycardia is still present.

In infants the attacks may occur during the first weeks or months of life and may not be diagnosed promptly because the

---

FIG. 5.—Various forms of rapid heart action, all but the last having rates ranging from 130 to 178. Note phasic variations in first sound (S_1) due to respiration with normal tachycardia. Essentially constant intensity of S_1 in paroxysmal atrial tachycardia, atrial flutter and nodal tachycardia. Marked variations in the first sound in atrial fibrillation and ventricular tachycardia; no heart sounds in ventricular fibrillation. (Courtesy of Levine, S. A., and Harvey, W. P.: *Clinical Auscultation of the Heart* [Philadelphia: W. B. Saunders Company, 1959].)

rapid heart rate is often overlooked by the parents. Instead the child may present symptoms such as vomiting, irritability, a rapid respiratory rate, hepatomegaly and cardiomegaly. It is therefore important to be aware of the possibility of paroxysmal atrial tachycardia occurring in children and to institute prompt and effective therapy. In contrast to the situation in adults, carotid sinus pressure or other local measures for vagal stimulation are generally ineffective in infants and children.

Rhythm

A regular rhythm is a characteristic feature (Figs. 4, 5 and 6). Irregularity should be an immediate cue to look for another type of tachycardia.

First Heart Sound

The first heart sound is constant in intensity, changing no more than one might expect from the minor variations heard with normal respiration (Fig. 5).

Carotid Sinus Pressure

This maneuver is of great importance in the diagnosis and treatment of paroxysmal atrial tachycardia. Either the attack is terminated with carotid sinus pressure (Figs. 4 and 6) or there is no effect. Most attacks, however, can be stopped by proper use of carotid sinus stimulation. Rarely one may note slight slowing without reversion of the tachycardia, but this represents an extreme exception to the rule. There is a difference of opinion as to the effectiveness of carotid sinus pressure in treatment of tachycardia: some observers state that only a minority, while others find that a majority, of patients with atrial tachycardia, revert to normal sinus rhythm after carotid sinus stimulation.

A few words on the technic of applying carotid sinus pressure appear appropriate (Fig. 7). Proper positioning of patient as well as physician is important. The patient lies flat on his back.

The physician places his left arm (or a pillow) under the patient's neck so that the head falls backward. The patient's head is then moved toward the opposite side, and the physician palpates for pulsations of the carotid artery at its bifurcation just below

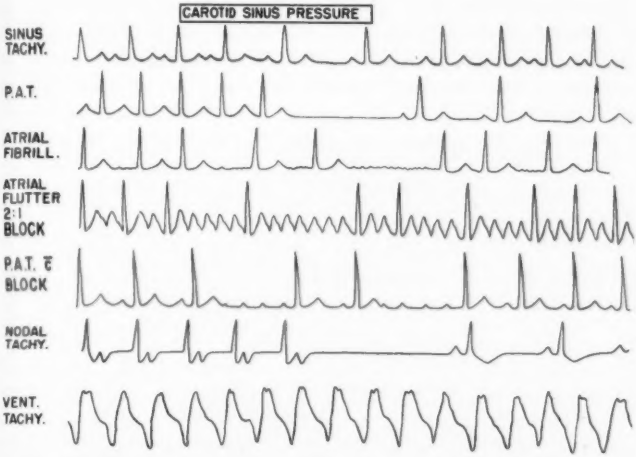

FIG. 6.—Composite of artist's sketch of effect of carotid sinus pressure on various tachycardias. *Upper strip:* Note gradual slowing and gradual return to former rate with normal sinus tachycardia. *Second strip:* Paroxysmal atrial tachycardia (PAT) abruptly stopped, followed by regular rhythm. *Third strip:* Atrial fibrillation. Rate originally irregular and rapid. Immediate slowing with irregular return to former rhythm. *Fourth strip:* Note prompt slowing of ventricles with irregular "jerky" return to original 2:1 flutter. The atria remain undisturbed. *Fifth strip:* Carotid pressure produces slowing with return to former rate. Note atrial waves easily identified during slowing. *Sixth strip:* Abrupt reversion from nodal rhythm to normal. *Seventh strip:* No effect whatsoever on ventricular tachycardia.

the angle of the jaw. If the carotid pulsation is not felt, response to carotid pressure is unlikely. Stimulation should not be attempted until the carotid artery pulsation is directly under the palpating finger or fingers. The stethoscope, as illustrated in Fig-

ure 7, is placed on the chest so that the physician is aware of the first moment when his efforts are effective, and additional unnecessary stimulation is not given. The right carotid sinus, being generally more effective than the left, should be chosen first, only one side at a time being stimulated. If the right side is ineffective, the left should then be tried, although as a rule if the right is ineffective the left is also. The fingers most useful for stimulating the carotid sinus have been the index, middle and fourth fingers, or the index and middle fingers. However, some physicians prefer the use of the thumb alone. As an initial maneuver

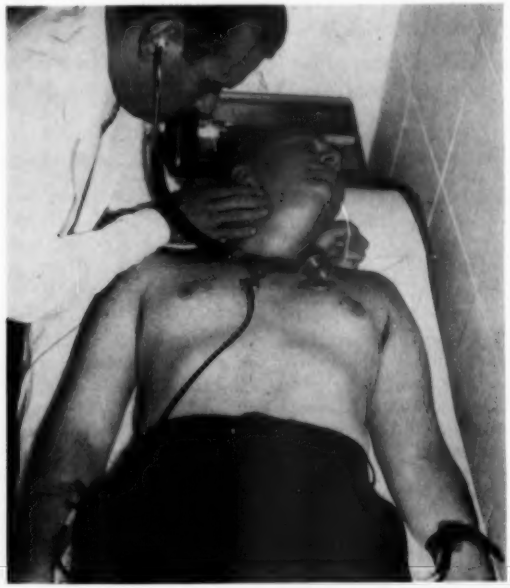

FIG. 7.—Technic of carotid sinus stimulation. The index and middle fingers are placed just below the angle of the left jaw over the carotid artery. The physician, listening with stethoscope, stops at the first response to stimulation. The electrocardiogram is recording the events, lead V_1 being used in this instance.

it is wise to press gently or moderately to see if the patient is abnormally sensitive. In some cases, when the carotid sinus is tested for hypersensitivity, lighter to moderate pressure is found to be sufficient. In the case of paroxysmal atrial tachycardia, however, many failures to respond are apparently related to the fact that too little pressure is exerted over the carotid sinus. Firm pressure is generally necessary—enough to indent a tennis ball with one's palpating finger. Pressure is applied with a massaging action for several seconds at a time, releasing for several seconds and then reapplying. This generally causes discomfort to the patient. Sometimes a series of five or six such intermittent stimulations are necessary before reversion abruptly takes place. A few useful "tricks" may be used, such as having the patient take a deep breath, hold it and strain at the moment that carotid sinus stimulation is started. A sudden change from a regular rapid rate to a slower normal rhythm is the type of response characteristic of paroxysmal atrial tachycardia or paroxysmal nodal tachycardia.

A few words of caution concerning the use of carotid sinus pressure: A cerebral vascular accident was observed in an elderly man with a history of cerebral vascular disease and a previous cerebral accident. Coincident with carotid sinus stimulation, a convulsive episode occurred and hemiplegia resulted. There is evidence that pressure on the carotid sinus may dislodge some atherosclerotic material which will embolize to the brain. It is also apparent that the decreased cerebral arterial flow resulting from occlusion of the carotid artery predisposes to complications in the susceptible patient. If possible, therefore, one should avoid carotid artery stimulation in people with known cerebral vascular disease. Prolonged carotid massage, ranging from 15 seconds to several minutes should also be avoided, for death has been reported as a complication from prolonged stimulation. Intermittent pressure for several seconds with release, repeating in a series of stimulations, has proved quite effective. Precautions such as listening with the stethoscope, plus electrocardiographic documentation, are further safeguards to cease stimulation immediately when a response has occurred.

Patients sometimes learn various maneuvers that may stop the attacks. These include taking a deep breath, holding it and straining; bending over and putting the head between the legs; lying across a bed with the head tilted toward the floor while holding the breath in deep inspiration and straining; producing gagging or vomiting by inserting a finger in the throat; drinking ice water; and applying pressure over the eyeball. Although some patients attempt to massage their own carotid sinus or eyeball, this practice should be discouraged, since this should only be done under proper medical supervision. As far as ocular pressure is concerned, this is largely ineffective. The physician is rightly hesitant about using enough force over the eyeballs to produce sufficient vagal stimulation; it generally induces significant pain over the eye and the possibility of permanent damage. Another "trick" which seems to have some value is placing the patient's hands and wrists in cold water. In other words, a cold pressor test is performed which theoretically and actually does cause some blood pressure elevation of a mild to moderate degree in some patients. At the same time carotid sinus pressure is repeated in combination with holding the breath in inspiration. Sometimes this has been successful when previous efforts have failed.

The practicing physician will encounter most of these cases in his office or in the patient's home. Local measures designed for carotid sinus stimulation are usually quite effective, and for this reason most patients do not require hospitalization. Whenever possible, it is best to have electrocardiographic documentation before, during and after cessation of the attack. A continuous electrocardiogram while carotid sinus pressure is being applied is strongly recommended, for it may be the only means of deciphering the arrhythmia in question.

In some cases, despite all of the maneuvers listed above, reversion to normal rhythm does not occur. In such instances drugs are required, and fortunately there is generally good success resulting from their use. A variety of drugs are available, and the drug (or drugs) of choice depend on the clinical situation and also on individual preference and experience. Since many people with paroxysmal atrial tachycardia are basically nervous and

tense, there may be considerable anxiety associated with the episode of tachycardia. Reassurance should be given and a sedative such as sodium pentobarbital or sodium secobarbital, 0.1 Gm., may be given. Digitalis has proved to be highly successful in terminating these episodes. If the patient has not had digitalis before—and most of them have not, since these attacks commonly occur in the absence of heart disease—a rapidly acting digitalis preparation such as lanatoside-C 0.8–1.6 mg. intravenously over a period of approximately 5 minutes is often successful. We prefer to give 0.8–1 mg. as the initial dose and, if necessary, additional lanatoside-C in increments of 0.2 mg. or 0.4 mg. every 30 minutes to 1 hour. Usually there is a period of 20–45 minutes before the drug causes reversion to normal sinus rhythm. If spontaneous reversion does not take place after an interval of approximately 30 minutes, one should repeat the simpler local measures, producing vagal stimulation and also carotid artery pressure. If the carotid sinus was insensitive before digitalization, it may now be quite sensitive and revert the tachycardia. Other digitalis preparations, such as digoxin, again given slowly by the intravenous route, will be just as effective. An initial dose of 1 mg. may suffice; if not, 0.25–0.5 mg. in increments of 0.25 mg. may be given at intervals of 30 minutes to 2 hours for 3 or 4 doses, repeating local measures, including carotid sinus stimulation, if reversion has not taken place before each additional dose of digitalis. Digitoxin 0.6–1.2 mg. orally or intravenously has also proved safe and effective. Digitalis is the drug of choice for initial treatment of paroxysmal atrial tachycardia if there are indications of impending congestive heart failure, particularly in an elderly patient or in any patient with heart disease predisposing to development of congestive heart failure.

Vasopressor drugs are likewise effective in treatment of paroxysmal atrial tachycardia. Phenylephrine in dosages up to 0.5 mg., diluted in a syringe to 10 cc. in 5% dextrose in water, may be given intravenously, slowly over several minutes. Frequent blood pressure determinations should also be made during the procedure, and monitoring with stethoscope and electrocardiogram is advocated. As described under treatment with digitalis prepa-

rations, local measures for stimulation of the carotid sinus should be repeated if there is not spontaneous reversion, particularly if the blood pressure has been elevated by administration of the drug. The success of vasopressor drugs is probably the result of reflex stimulation of the carotid sinus coincident with elevation of blood pressure. When a vasopressor is given by the intravenous route, injections should be stopped at any point where reversion takes place, even though only a small portion of the drug has been given. The use of a vasopressor drug is particularly indicated as the first drug of choice in any shocklike state or if significant hypotension is associated with the tachycardia. Other vasopressors such as levarterenol (Levophed),/ampule (4 cc. of 0.2% solution of levarterenol bitartrate), diluted in a liter of 5% dextrose in water may be given intravenously, using blood pressure as a guide and repeating the same steps as outlined above.

Prostigmine 0.5–1 mg. intramuscularly is often successful after an interval of 20–30 minutes. If spontaneous reversion has not occurred by this time, however, simple or local measures producing vagal stimulation, including carotid sinus pressure, should be repeated. The carotid sinus may now be sensitive, resulting in reversion of the tachycardia to normal sinus rhythm. Prostigmine should be avoided in asthmatic patients and in those with an allergic background. As a precaution when using prostigmine, the physician should always have ready, if necessary, a syringe of atropine (1–2 mg.) for intravenous use to counteract any severe vagal reaction, particularly bronchospasm. If any untoward symptoms do occur, the application of a tourniquet about the site of the intramuscular or subcutaneous injection may reduce the absorption of the injected drug.

At times it may be necessary to use any combination of the drugs (digitalis, prostigmine and vasopressors).

In our experience the local measures, particularly carotid sinus stimulation, will terminate most attacks of paroxysmal atrial tachycardia. Those cases that do not respond will generally do so by the use of digitalis, vasopressors or prostigmine. If a patient is still resistant after these treatments, the next step is

use of oral quinidine, unless an unusually urgent, grave situation requires more heroic measures.

The patient who has had only one episode of paroxysmal atrial tachycardia, or who has had recurrences at infrequent intervals of every few years, does not need daily prophylactic medication. On the other hand, for the patient whose paroxysms are recurring daily, or several times a week, prevention becomes an important part of treatment. Several steps are used in prevention, including reassurance, sedation, digitalis and quinidine. Sometimes the simple use of reassurance plus sedation proves beneficial, but if the attacks are recurring with any significant frequency, this generally does not suffice. The next step, while continuing sedation, is to digitalize the patient in the usual manner (e.g., as one would use digitalization for cardiac decompensation).

It is roughly estimated that approximately one out of four patients are benefited by digitalization. If they are not, then digitalis is discontinued—sedation may or may not be continued, depending on the patient—and oral quinidine is started. This has been the most effective drug, usually in a dose of 0.3 Gm. at approximate intervals of 6 hours. If quinidine sulfate 0.3 Gm. is given after each meal and at bedtime, there appears to be less likelihood of diarrhea or gastrointestinal distress in most patients. Occasionally a smaller dose of 0.2 Gm. 3 times a day is effective, but other patients require more frequent and larger doses in order to have adequate protection against recurrences. Occasionally the patient will be well controlled during the day but be awakened from sleep by tachycardia. For such a patient the preparation quinidine polygalacturonate 0.3 Gm. at bedtime with its theoretically longer action may be effective. In case it is not, and a larger dose at bedtime also fails, the patient may find it necessary to set an alarm clock to take an additional tablet of quinidine in the middle of the night in order to keep an effective level throughout his sleeping as well as his waking hours.

Procaine amide may also be used as a prophylactic in doses of 250–500 mg. every 4–6 hours. This drug has been especially

useful for those patients unable to tolerate quinidine in the usual dosage.

PAROXYSMAL ATRIAL FIBRILLATION

This represents one of the more common, as well as important, tachycardias.

Rate and Rhythm

This tachycardia is not difficult to recognize, due to its rapid irregular heart rate. The rate varies between 100 and 200 (Figs. 5 and 8), although at times rates of over 200 may be noted. It is frequently seen in conditions such as rheumatic valvular disease, particularly with mitral stenosis and/or insufficiency, hypertensive heart disease, arteriosclerotic heart disease and thyrotoxicosis. It may develop coincident with infections such as pneumonia or during anesthesia or surgery for various conditions. Paroxysmal atrial fibrillation may occur in a patient with no other evidence of heart disease; such patients are known as normal or idiopathic fibrillators. An unusual, although documented, complication of such patients is the development of congestive heart failure associated with an uncontrolled tachycardia, particularly if reversion has not taken place promptly. At times alcoholic beverages,

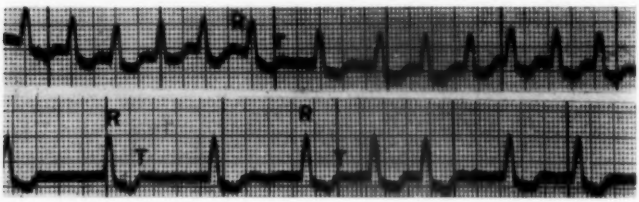

Fig. 8.—Electrocardiogram showing atrial fibrillation. Note faster grossly irregular rate in upper strip and slower irregular ventricular rate in lower strip.

particularly in excess, appear to be a precipitating agent in paroxysmal atrial fibrillation.

This tachycardia may come in short paroxysms, lasting a few minutes, hours or days, but it often remains permanently unless medication is given to cause reversion to normal rhythm. The heart rate is totally irregular with rapid beats interspaced with slower beats, producing no dominant recurring pattern. The tachycardia may occur suddenly from a previous normal sinus rhythm; on the other hand, a heart previously fibrillating at a slow rate may, under periods of stress, infections or operations, become rapid. This abrupt change from a normal sinus rhythm to a rapid, grossly irregular, palpitating heart with a rate of 180–190 is generally readily appreciated by the patient and may cause apprehension and alarm. I had the personal experience of awakening one night from a dream and being immediately aware of a tumultuous pounding caused by a rapid irregular heart rate. This experience gave me a vivid illustration of what a patient may experience when first having this rhythm. It was a frightening experience, to say the least, and even after I realized what the rhythm was, an uncomfortable awareness of the atrial fibrillation coupled with a somewhat subdued feeling of anxiety remained until a sedative took effect, producing sleep. Quinidine sulfate was also taken, and on awakening the following morning, I found the rhythm was regular. I also learned, however, of some of the side effects of quinidine administration—diarrhea for the remainder of the day plus a feeling of undue exhaustion.

First Heart Sound

The first heart sound in tachycardia due to atrial fibrillation changes in intensity from slight to greatly accentuated (Fig. 5). There is no orderly change of the intensity of the first sound, but instead it occurs in a haphazard fashion. This changing first sound, coupled with the totally irregular ventricular beat, is a characteristic finding in atrial fibrillation.

Carotid Sinus Pressure

This may produce slowing of the rapid rate but does not cause reversion to normal sinus rhythm. The irregular rapid rate slows but remains irregular, and then returns to its former rapid rate (Fig. 6). This arrhythmia is generally one of the easier ones to recognize at the bedside, in view of the totally irregular beat.

At times the occurrence of a rapid paroxysm of atrial fibrillation may be associated with severe precordial distress of coronary insufficiency, which requires morphine or another narcotic for relief. Some patients are completely free of their anginal or coronary insufficiency pain except during these episodes of tachycardia. A patient illustrating this was a man of 60 seen during the past year for evaluation of recurring (once or twice per week) severe precordial pain, typical of coronary insufficiency and requiring 200 mg. meperidine for relief. He was incapacitated for one or two days with each episode. Between these episodes he felt perfectly well. His electrocardiogram showed normal sinus rhythm and nonspecific S-T and T wave changes but no evidence of myocardial infarct, old or recent. The history, plus the mimicking of rapid tachycardias with the hand over the precordium (Fig. 3), made it apparent that the severe pain was always associated with rapid irregular heart action, most likely paroxysms of rapid atrial fibrillation. He was then digitalized and several days later noted palpitation, but this time without pain. The electrocardiogram documented atrial fibrillation, but at a ventricular rate of approximately 110. The slower ventricular rate as a result of digitalis was not accompanied by coronary insufficiency, whereas the faster rate was. Spontaneous reversion to normal sinus rhythm occurred; he was then placed on maintenance quinidine 0.3 Gm. every 6 hours and has apparently been symptom-free for the past 9 months. In such patients with arteriosclerotic heart disease, treatment of coronary pain is dependent on prevention of the paroxysms of rapid heart action.

Electrocardiogram

The electrocardiogram (Figs. 5, 6 and 8) shows the grossly irreg-

ular ventricular rate with fibrillary waves rather than P waves of atrial contraction. Whether the ventricular rate is very rapid or slightly so, the irregular beat having no definite recurring pattern is typical.

Treatment

As a rule, digitalis is the drug most effective for control of this tachycardia. Any of the digitalis preparations, such as digitalis leaf, digoxin, digitoxin or lanatoside-C, may be used according to the preferences of the physician. The action of digitalis is generally prompt and specific in slowing ventricular rate. One can judge digitalis administration by using the ventricular rate as a guide to the amount of digitalis needed. In some cases an underlying complicating process such as thyrotoxicosis, infections (e.g., pneumonia), myocarditis or pulmonary emboli, must also be treated. In the uncomplicated case, digitalis may be administered until the ventricular rate is in the approximate range of 60–80. For example, a previous rate of 170, after 0.8–1 mg. lanatoside-C is given intravenously, might be reduced to 110 or 120 within 30 minutes to 1 hour. Thereafter gradual increments of 0.2–0.4 mg. may show that after a total dose of 1.4–1.8 mg. has been given, the ventricular rate is 70 or 80. At this point I prefer to continue a maintenance dose of digitalis rather than discontinuing it if reversion to normal sinus rhythm is contemplated.

In some cases, particularly those unrelated to rheumatic mitral valvular disease, reversion to normal sinus rhythm may spontaneously occur. In approximately three fourths of the cases, however, quinidine is necessary to effect reversion. This may be accomplished by continuing the maintenance digitalis and giving quinidine according to one of the schedules to be described (Tables 1 and 2). On occasion paroxysmal atrial fibrillation may occur in association with hypotension or shocklike states. Treatment of the underlying cause is necessary, including the administration of a vasopressor such as levarterenol or metaraminol. If effective in elevating blood pressure, the drug may also

accomplish reversion of the fibrillation to normal sinus rhythm. Sometimes a spontaneous paroxysm of rapid atrial fibrillation may be treated with quinidine alone plus sedation, although as a rule I prefer to give the sedation and digitalis first in order to insure a slower fibrillating rate before starting quinidine therapy. Once rapid atrial fibrillation has been slowed, reversion is generally attempted. The hardest patients to revert are those with mitral valvular disease, particularly those having significant mitral insufficiency with large or giant atria; they also represent the most difficult patients to keep in a regular rhythm. At some point in the course of treatment, the decision is generally made to let the patient remain in atrial fibrillation, the ventricular rate controlled with maintenance digitalis. For those patients not having chronic rheumatic mitral valvular disease (e.g., the patient with arteriosclerotic or hypertensive heart disease having frequent recurrences of atrial fibrillation) prevention of future attacks may be accomplished by a maintenance dose of digitalis, but more often the additional use of quinidine is necessary. Particularly if there is any evidence of underlying heart disease where congestive failure has been a problem in the past, or if there is likelihood that this may follow a rapid rate, digitalis is indicated as maintenance therapy with the additional use of quinidine 0.2 or 0.3 Gm. several times daily.

In a discussion of the treatment of atrial fibrillation, it is particularly pertinent to mention some of the methods currently used in administering quinidine for reversion to normal sinus rhythm. The use of oral quinidine is preferable when the clinical situation indicates no urgency. In attempting to revert a patient with atrial fibrillation, a method advocated by Levine has proved safe, conservative and effective. As illustrated in Table 1, increasing increments are given over a period of several days. Doses are given 3 times a day, and each dose of quinidine is greater than the succeeding one. The first daily dose is generally given after the morning meal; the second dose is given in the early afternoon (also after a meal); and the last dose is given at bedtime. Before receiving each dose of quinidine, the patient is checked by a physician as to his clinical state. If indicated, an

electrocardiogram may be recorded before the administration of the next dose. It is emphasized that the patient should be checked by a physician before each additional dose of quinidine. On such a schedule, the patient is given a sedative to insure a good night's sleep, because not infrequently patients will revert to normal sinus rhythm during their sleeping period. Daytime sedation in addition to quinidine also appears of value. On the succeeding day a larger single dose than the one given the night before is administered; each succeeding dose thereafter is gradually in-

TABLE 1.—LEVINE'S METHOD OF QUINIDINE ADMINISTRATION
(Doses in Grams)

	1ST DAY	2D DAY	3D DAY	4TH DAY
Morning:	0.2	0.5	0.8	1.2
Early Afternoon:	0.3	0.6	0.9	1.4
Bedtime:	0.4	0.7	1.0	1.5

creased in amount; if the patient has not reverted after the last day's treatment consisting of 1.2, 1.4 and 1.5 Gm., this method is discontinued. Another method may be tried after giving the patient a rest of several days.

Although an occasional patient may revert on the initial small doses of the first day's schedule, in our experience most revert to normal sinus rhythm between dosages 0.6 and 1.2 Gm. At any signs of unusual toxicity—either by clinical or by electrocardiographic control—quinidine is discontinued. This trial at reversion should be performed in the hospital where close supervision is possible. When reversion occurs, the patient is given maintenance doses of 0.2 or 0.3 Gm. quinidine after each meal and at bedtime. We have found that the use of oral quinidine given after meals has lessened the likelihood of diarrhea and/or gastrointestinal upset. Also helpful in alleviating the incidence of toxic gastrointestinal side effects from quinidine administration has been the use of aluminum hydroxide gel between meals and at bedtime. This has proved particularly useful when diarrhea has been a complicating side effect, since aluminum hydroxide has a tendency to bring

about constipation, compared with some of the other antacids to which magnesium has been added to counteract this tendency.

Other methods of quinidine administration are designed to give doses at more frequent intervals over a day's period. This may accomplish the desired effect more rapidly. Doses generally varying from 0.2 to 0.6 Gm. are given at specified intervals for a specified number of doses. One method of this type is illustrated in Table 2, in which smaller single doses are given at intervals

TABLE 2.—Rapid Method of Quinidine Administration
(Doses [Gm.] at Intervals of 2–6 Hours)

1st Day	2d Day	3d Day	4th Day	5th Day	6th Day
0.2	0.3	0.4	0.5	0.6	0.7
0.2	0.3	0.4	0.5	0.6	0.7
0.2	0.3	0.4	0.5	0.6	0.7
0.2	0.3	0.4	0.5	0.6	0.7
0.2	0.3	0.4	0.5	0.6	0.7
0.2	0.3	0.4	0.5	0.6	0.7

varying from 2 to 6 hours. There are a number of variations of this method of quinidine administration which depend on the preference of the individual physician in addition to the clinical situation. As a practical rule, a total day's dosage of quinidine of 3–3.5 Gm. suffices. Additional doses should be used with caution, since toxicity from quinidine itself may then occur. As emphasized by Sokolow, serum concentrations of quinidine may be helpful in evaluating administration of this drug. If a serum concentration over 8 mg./L. has been reached, toxic effects may ensue. The physician should familiarize himself with various methods of quinidine administration, since at times the rapid method (Table 2) may be more successful than the slower method (Table 1). On the other hand, we have seen successful use of Levine's method, where other methods had failed.

When reversion has been unsuccessful, it is wise to review the situation to see if any underlying factors are working against the attempt at reversion with quinidine, such as thyrotoxicosis, fever,

infections, recurrent pulmonary emboli, immediate postoperative period (e.g., after mitral commissurotomy) or chronic rheumatic heart disease with predominant mitral insufficiency and giant left atrium. The latter group of patients appears most difficult to revert and keep reverted. At times, however, attempts at reversion in such patients are justified because, with normal sinus rhythm, cardiac decompensation may be fairly well controlled, while the patient with atrial fibrillation may have uncontrolled heart failure. It appears probable that most patients who have started to fibrillate recently—within hours, days or several weeks —are easier to revert than those who have had long-standing fibrillation. However, even in some patients with long-standing atrial fibrillation, reversion may be accomplished quite easily.

A word of caution should be given concerning patients starting to fibrillate in the period after mitral commissurotomy. This is not an unusual complication; a normal sinus rhythm is present before mitral commissurotomy, but during the immediate postoperative period—or at the time of surgery—atrial fibrillation occurs. Quinidine may be given in smaller doses, and reversion may be successful, but use of the full schedule is to be discouraged, as toxic effects from quinidine administration are much more likely at this time. Several weeks or months after postoperative hospitalization, reversion may be more safely carried out.

The patient with atrial fibrillation who is having symptoms of cardiac decompensation—other than the chronic fibrillator with rheumatic mitral disease—generally warrants, after a total clinical assessment of the case, a trial at conversion. Most patients are benefited if the rhythm can be made regular. The patient who continues to fibrillate after thyrotoxicosis should also be given the benefit of quinidine as an attempt at reversion. It goes without saying that a patient with atrial fibrillation and thyrotoxicosis should have his thyrotoxicosis treated first; spontaneous reversion to normal sinus rhythm may then occur. It is estimated, however, that approximately half of these patients continue in atrial fibrillation. It should be remembered to give quinidine to revert these cases, because generally this can be

accomplished with ease. It is also important to effect reversion in the normal or idiopathic fibrillator and particularly in the younger patient with arteriosclerotic or hypertensive heart disease. During reversion attempts, electrocardiographic control is important because the use of quinidine or other antiarrhythmic drugs is not without danger, and may in themselves produce untoward effects; however, used wisely and conservatively they are most beneficial. Although this discussion has emphasized the use of quinidine predominantly in the reversion of atrial fibrillation, procaine amide may also be of value. We have found, as others have, that the combined use of quinidine and procaine amide in selected cases has been of benefit.

ATRIAL FLUTTER

Rate

In this condition the atria are contracting at a rate between 250 and 350; they actually contract, but the atrioventricular node does not conduct all of the impulses to the ventricles. In the untreated case of atrial flutter, there is usually a 2:1 response, the ventricles responding to every other atrial contraction. Therefore, the average patient with atrial flutter who has received no medication has a regular ventricular rate one-half that of the atrial rate (e.g., ventricular rate of 160, atrial rate of 320). In rare instances all of the atrial contractions are conducted, resulting in a 1:1 response. If the patient has already received medication (digitalis and/or quinidine), varying degrees of A.V. block are common.

Atrial flutter is more likely to be persistent, lasting for days, weeks or even longer unless treatment is instituted. Persistent atrial flutter is more likely to be associated with organic heart disease, such as rheumatic, hypertensive or arteriosclerotic heart disease. However, it may occur in transient episodes. Occasionally it is seen in a person with an otherwise normal heart, and the same person may sometimes present with paroxysmal atrial fibrillation. I had the opportunity of following such a patient for a number of years. He had a documented history of approxi-

mately 40 years of intermittent paroxysmal heart action, either atrial fibrillation or flutter; between attacks he had a normal heart, as far as could be determined by the usual clinical criteria.

The sudden onset of a rapid regular rate might be confused at times with paroxysmal atrial tachycardia; at other times a rapid ventricular rate with varying degrees of A.V. block simulates atrial fibrillation. The bedside diagnosis of atrial flutter has generally been regarded as difficult or unlikely, and it has been thought that this rhythm will be recognized initially only if an electrocardiogram has been taken. By paying close attention to the combination of the rate, first heart sound and response to carotid pressure, however, diagnosis may be made with a surprising degree of accuracy.

First Heart Sound

In the untreated case of flutter, the first heart sound is generally constant, not changing in intensity (Fig. 5), but once treatment is instituted, a changing intensity of the first heart sound is commonly observed if one pays attention to this auscultatory point.

Carotid Sinus Stimulation

Atrial flutter may be suspected if carotid sinus pressure produces slowing of the ventricular rate, followed by an irregular return to its original rate (Fig. 6). The "jerky" return characteristic of atrial flutter, in contrast to a gradual or smooth return generally seen with normal sinus tachycardia, results from the varying degrees of A.V. block produced by the carotid pressure before the return to the former ventricular rate. Occasionally, the slowing may result in a halving of the ventricular rate, and in that case the degree of A.V. block is doubled (e.g., from 2:1 to 4:1). Ventricular rate may continue at a slower rate, and if this occurs, the likelihood of atrial flutter is strong. Generally, however, the rate will return to its original, more rapid rate after periods of time varying from seconds to minutes. It is important to emphasize that it is not merely the slowing in such a

case, but the change of the ventricular rate to one-half its original rate. This would distinguish it from paroxysmal atrial tachycardia in which reversion to normal sinus rhythm would be unlikely to be exactly one-half of the rate of the tachycardia. At other times the patient may have atrial flutter with a constant degree of A.V. block, such as 4:1, and after exercise the rate may double, signifying a change in A.V. block from 4:1 to 2:1. A simple diagnostic test is to have the patient take a deep breath, hold it and strain. This may cause a slowing of the ventricular rate with the characteristic jerky return, or a halving of the rate similar to the effect of carotid sinus pressure. It would be unusual if any tachycardia responded to this maneuver other than atrial flutter (except, at times, paroxysmal atrial tachycardia with block).

Electrocardiogram

When possible, the electrocardiogram should be taken in all types of tachycardias. In atrial flutter, characteristic findings of notched, saw-tooth type of flutter waves may be readily identified, particularly in leads II and III (Fig. 6). The underlying arrhythmia may also be particularly well identified in lead V_1 of the electrocardiogram. A strong plea is made that for correct identification of an arrhythmia the electrocardiogram be recorded while carotid sinus stimulation is being applied (Fig. 7), using the technic already described for carotid sinus pressure, listening with stethoscope to detect the moment of response to carotid pressure. At times atrial activity is not readily apparent from the electrocardiogram when there is a 2:1 response, because one of the P waves may be buried in the T wave; only on carotid sinus stimulation is the underlying mechanism disclosed, enabling the observer to make the correct diagnosis.

Treatment

The best treatment of atrial flutter is the use of digitalis. In the untreated case with rapid ventricular rate, digitalis is gener-

ally effective in promptly increasing the degree of A.V. block, thereby reducing the ventricular rate. The use of a rapidly acting digitalis preparation, such as lanatoside-C intravenously in doses of 0.8 or 1 mg. initially, may effect prompt slowing in a period of 15–45 minutes. Similar to its use in atrial fibrillation, lanatoside-C in additional doses may be given in increments until the degree of A.V. block is high enough to result in slowing and irregularity of the ventricular rate. It is not unusual that a full digitalizing dose of 1.6–1-8 mg. of lanatoside-C will be given, producing slowing of the heart rate without changing atrial flutter to fibrillation nor reverting the rhythm to normal sinus rhythm. One would hope to have the flutter change into fibrillation, and larger doses of digitalis are often required to accomplish this. Occasionally even twice the digitalizing dose, given in increments over a period of several days, may be necessary before atrial fibrillation or reversion to normal sinus rhythm takes place. At other times even higher doses of digitalis are required. At any signs of toxicity digitalis must be discontinued, but it has been a frequent clinical observation that patients with atrial flutter tolerate larger doses surprisingly well.

If atrial fibrillation occurs, our practice has been to continue the patient on a maintenance digitalis dosage, and then on a subsequent day—1 or 2 days later—to start quinidine administration. Although some physicians advocate discontinuing digitalis at this point, we have not found this necessary nor advantageous and prefer to continue a maintenance dose. Quinidine is administered in a schedule (Table 1 or 2), similar to the method used in reversion of atrial fibrillation. If reversion to normal sinus rhythm is accomplished, the patient is maintained on quinidine, usually 0.3 Gm. 3 or 4 times daily. At times, as discussed previously, an increased maintenance dose of quinidine is necessary for prevention of recurrent attacks. In case reversion to normal sinus rhythm cannot be effected, the patient is kept on maintenance digitalis for control of his ventricular rate. It is desirable, if possible, at least to change the rhythm from flutter to atrial fibrillation, if not to normal sinus rhythm.

At times one may examine a patient with atrial flutter having

a higher degree of A.V. block and a ventricular rate of 70 or 80. The question is then asked: "Why try to convert this to atrial fibrillation? Why is the flutter not just as safe as fibrillation with the same ventricular rate?"

Coincident with various stimuli, such as infections, undue effort, emotions, etc., the patient with atrial flutter may suddenly double his rate (e.g., go from 80 to 160). In a patient with organic heart disease, the rise may be followed by untoward symptoms of congestive heart failure or the coronary insufficiency type of pain. The same patient, if fibrillating, may under the same circumstances increase his rate from 80 to a range of 110–120, without the symptoms described. It is worthy of emphasis that the usual patient with untreated flutter having 2:1 block is better treated first with digitalis and subsequently by quinidine administration, if necessary. We prefer that quinidine not be the first drug used in treatment of atrial flutter, but certainly it has an important role in subsequent treatment. The use of quinidine first—without digitalis—is probably more likely to predispose to the occurrence of a 1:1 response, often with alarming consequences and symptoms.

PAROXYSMAL ATRIAL TACHYCARDIA WITH BLOCK

During the past 10 or 15 years paroxysmal atrial tachycardia with block has been well emphasized, particularly in reference to the fact that it often represents a manifestation of digitalis toxicity. The publications of Lown and Levine have emphasized this tachycardia and its relation to digitalis-potassium imbalance and have stressed the necessity of treating this rhythm with potassium and/or procaine amide. There is a misconception in the minds of some, however, that the presence of paroxysmal atrial tachycardia with block *always* indicates digitalis toxicity. In our institution approximately two-thirds of the patients have had this tachycardia as a manifestation of digitalis intoxication, and in the other one-third it was unrelated to digitalis or potassium depletion. Ironically enough, the type of treatment given those who have never received digitalis has been the ad-

ministration of digitalis, similar to the treatment described for atrial flutter. This tachycardia, though designated paroxysmal atrial tachycardia with block has some features of paroxysmal atrial tachycardia, but there are major differences. At the same time it has some of the features of atrial flutter, but also with differences; it is not a tachycardia entirely characteristic of either atrial flutter or paroxysmal atrial tachycardia. As in atrial flutter, varying degrees of A.V. block may be present.

Electrocardiogram

Instead of the notched sawtooth type of flutter waves, the P waves are smaller, having a flattened isoelectric line (Figs. 6 and 9).

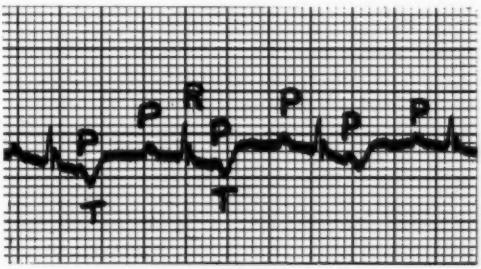

Fig. 9.—Paroxysmal atrial tachycardia with block. Note that one P wave coincides with T wave. Unless carefully analyzed, a P wave such as this is sometimes overlooked and the rhythm is mistaken for normal sinus. Atrial rate is 166.

First Heart Sound

Although similar to that heard in atrial flutter, particularly if there is a constant A.V. block, the first heart sound here may change when there is a varying degree of block. The ventricular rate is generally slower than in flutter, and the atrial rate in this condition generally ranges between 150 and 250, although at times it may be slower or faster.

Carotid Sinus Pressure

This form of tachycardia resembles flutter in that the ventricular rate may be slowed and a "jerky" type of return to the former rate may occur. Paroxysmal atrial tachycardia with block differs from atrial flutter in that it is more difficult to treat or to revert to normal sinus rhythm. It is also much more difficult to revert than paroxysmal atrial tachycardia. In some patients this abnormal rhythm has been known to exist for months or even years. At times the diagnosis was unsuspected even though electrocardiograms had been taken, because a P wave was buried in the preceding T wave. It follows, therefore, that in treatment those measures aimed at carotid sinus stimulation, as in paroxysmal atrial tachycardia, will be ineffective. Except for aid in establishing the diagnosis, carotid sinus pressure, as with atrial flutter, has no place in the treatment of this tachycardia.

An interesting feature of paroxysmal atrial tachycardia with block is that this has been a common manifestation of digitalis toxicity, and there appears to be a definite relationship between manifestations of digitalis intoxication and potassium imbalance. In a number of cases, signs of digitalis toxicity have been present after diuresis if there has been a significant loss of potassium, even though there is no history of increased digitalis medication. The administration of potassium may be effective in eliminating this arrhythmia.

Treatment

Treatment of paroxysmal atrial tachycardia with block involves two main groups of patients: those already receiving digitalis, in whom this tachycardia represents a manifestation of digitalis toxicity; and those patients who have never received digitalis. In the first group, if potassium imbalance is present, patients are best treated by discontinuing the digitalis and administering potassium, provided the urinary output is normal and no significant renal insufficiency is demonstrable. If the situation is more urgent as far as the tachycardia is concerned, 40 mEq.

potassium chloride in 500 ml. of 5% dextrose in water may be slowly given intravenously over a period of approximately 1 hour. If at the end of the infusion, the atrial rate is slowed but reversion has not yet been accomplished, additional potassium may be given. If at any time during the intravenous administration, the revision occurs, the potassium is stopped. Procaine amide is also useful, and at times may be given in conjunction with potassium in dosages of 1 Gm. orally and additional doses of 0.5 Gm. every 3–6 hours. Procaine amide may also be given intravenously—according to the technic described under paroxysmal ventricular tachycardia—in which 50–100 mg. are given every 2 minutes. The procaine amide is discontinued if there is a fall in blood pressure. After elevation of blood pressure with a vasopressor, procaine amide may be slowly resumed. At times potassium and procaine amide are given simultaneously, particularly in a more serious situation, and with a more rapid tachycardia. If the situation does not appear urgent, the potassium may be given orally—4–5 Gm. in cold fruit juice, followed several hours later, if necessary, by one-half this dose. It is important to emphasize that electrocardiographic monitoring should be performed during the treatment of this arrhythmia and its response to medication.

Although most patients with paroxysmal atrial tachycardia with block represent a manifestation of digitalis toxicity and the potassium relationship as described, a number varying from one-fifth to approximately one-third of the patients have never received digitalis treatment, and treatment of this group with potassium and/or procaine amide will be unsuccessful. Ironically enough, the treatment in these cases is similar to that of atrial flutter where digitalis—often in larger doses—is necessary, and the method of treatment is essentially that outlined under the treatment of atrial flutter. Before giving digitalis to a patient with paroxysmal atrial tachycardia with block, however, it is of utmost importance to double-check by every possible means to make sure that the patient is not receiving digitalis without the knowledge of himself or his physician.

NODAL TACHYCARDIA

This tachycardia is the least common of the tachycardias outlined in this discussion and has characteristics similar to those discussed under paroxysmal atrial tachycardia. The *rate* is quite variable from a slight elevation to more rapid, similar to that of paroxysmal atrial tachycardia. The *rhythm* is regular, and the first heart sound does not change in intensity. *Response to carotid pressure* (Fig. 6) may be similar to that in paroxysmal atrial tachycardia, in which either reversion to normal sinus rhythm occurs or no effect is produced. Nodal tachycardia can often be suspected by noting prominent canon waves of the jugular venous pulse. The *electrocardiogram* may show the P wave coming after the QRS (Fig. 5), although at times it may just precede or occur in conjunction with the QRS. The P wave may be inverted or altered in form, or at other times it is upright.

VENTRICULAR TACHYCARDIA

Paroxysmal ventricular tachycardia is the most serious of the tachycardias. When present it generally indicates a serious underlying heart disease, such as coronary artery disease, with or without myocardial infarction, primary myocardial disease (or myocarditis) or manifestations of other types of heart disease, such as rheumatic or hypertensive. An abnormal focus in the ventricles produces a series of ectopic beats resulting in a rapid tachycardia. Ventricular tachycardia may last only a few seconds or minutes but at times will last hours or days.

Ventricular Rate

Rate usually ranges between 150 to 250. The onset may be abrupt and similar in rate to that seen in paroxysmal atrial tachycardia, but it differs in many respects. If one listens carefully for the ventricular rate, or measures this on the electrocardiogram, a slight irregularity may be noted. This variation may be no more than several hundredths of a second but may be appreciated by

auscultation. At faster rates the ear can readily detect slight alterations in regular rhythm, such as .03 or .02 second. At other times the ventricular rate may be regular, not showing these slight variations.

First Heart Sound

Characteristically these sounds vary in intensity, sometimes loud, sometimes faint (Figs. 5 and 10). This is due to the changing P-R relationship. The rates of the ventricles and atria are dissoci-

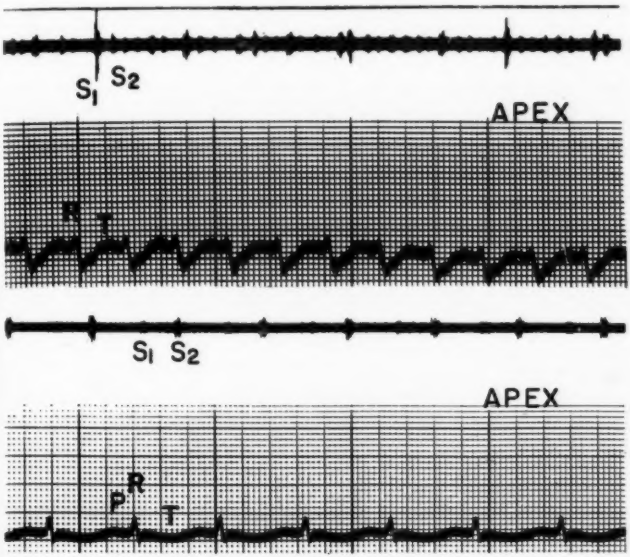

Fig. 10.—Multiple sounds in ventricular tachycardia, in man, 58, several weeks after acute myocardial infarction. Note changing intensity of first sound (S_1) in ventricular tachycardia (*upper tracing*) and absence after reversion to normal sinus rhythm with quinidine (*lower tracing*). S_2 = second sound.

ated, with the ventricles beating at a faster rate than the atria. When the atrial contraction (or P wave) just precedes the ventricular contraction, the first heart sound will be accentuated. If the P-R interval is prolonged to a certain degree, the first heart sound will be faint. The change in intensity of the first heart sound represents a valuable clue which may first alert the physician to the possibility of ventricular tachycardia. Further evidence of the relation of the P-R interval to the intensity of the first heart sound is the fact that patients with ventricular tachycardia who also have atrial fibrillation have not shown this change in intensity of the first heart sound; there was no atrial contraction and therefore no changing P-R relationship.

In addition to this important auscultatory sign in paroxysmal ventricular tachycardia, another auscultatory clue has been the hearing of multiple sounds (Figs. 5 and 10). These are generally of low frequency and are best heard at the apex. I have been impressed with the clinical observation over a number of years that ventricular tachycardia usually sounds different from other tachycardias. On careful analysis it has become apparent that additional sounds of ventricular tachycardia are a major cause of this auscultatory peculiarity. These sounds are apparently produced by a wide splitting of the first and second heart sounds (as with bundle branch block) plus gallop sounds. Because of the spread of the QRS complex, there is wide splitting of the first and second heart sounds; a ventricular diastolic gallop rhythm is frequently present, and an atrial gallop sound may also be heard. I have observed in some patients that a sound in systole may be heard in addition to the aforementioned sounds. These give rise to multiple staccato sounds which, combined with the changing intensity of the first sound and the slightly irregular ventricular rate, produce the characteristic auscultatory findings of ventricular tachycardia. These features, combined with other clinical aspects to be mentioned, aid in the prompt recognition and treatment of this arrhythmia, which is of such serious import. Most, although not all, patients with ventricular tachycardia show these auscultatory findings. Production of these sounds is directly related to a number of factors: widening of the QRS complex, duration of the

ventricular tachycardia, lack of cardiac reserve and the degree of cardiac decompensation.

Carotid Sinus Pressure

Sounds heard in other forms of tachycardia occasionally simulate those heard in ventricular tachycardia. This is particularly true of rapid atrial fibrillation with aberrant conduction, to be discussed shortly. Atrial flutter with bundle branch block and audible atrial sounds, and sinus tachycardia with bundle branch block, might also be confused with ventricular tachycardia. The slowing of heart rate by carotid sinus pressure, however, helps to identify these and to rule out ventricular tachycardia.

Carotid sinus stimulation is not effective in ventricular tachycardia (Fig. 6). We have never observed a case in which ventricular tachycardia was slowed or reverted with carotid sinus pressure, although one physician—an authority on arrhythmias—told me that he had observed two such instances. For all practical purposes, however, slowing of the tachycardia with carotid sinus pressure may be taken to indicate a tachycardia other than the paroxysmal ventricular form.

Aberrant conduction with a widened QRS complex plus the rapid tachycardia of atrial fibrillation may simulate ventricular tachycardia. In this instance, the rhythm may be rapid and irregular, with a changing intensity of the first heart sound. In such cases of aberrant conduction with atrial fibrillation, however, the rhythm is generally much more irregular. Instead of varying from .01 to .04 second, as with ventricular tachycardia, there might be variances of .08–1 second or even longer between beats, with rapid atrial fibrillation and aberrancy. The effect of carotid pressure is most useful, as slowing produced on carotid sinus pressure eliminates the possibility of ventricular tachycardia. It is again worthy of emphasis that the electrocardiogram be taken during carotid sinus stimulation to rule out the possibility of any slowing that might not be detected while the carotid massage is being carried out.

The *electrocardiogram* in ventricular tachycardia reveals the

abnormally widened QRS complex and A.V. dissociation with the P waves at a slower rate than the ventricles (Fig. 11). At times there is a retrograde conduction of the P waves, and in such cases the change in intensity of the first heart sound is not evident.

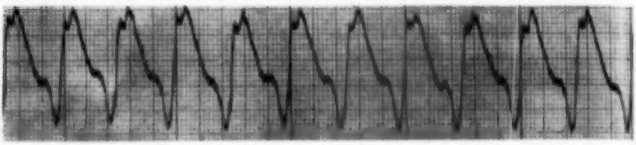

FIG. 11.—Electrocardiogram of ventricular tachycardia. Note very wide QRS complex and ventricular rate of 120. This patient with arteriosclerotic heart disease also had multiple sounds and a changing intensity of the first heart sound. Carotid sinus pressure produced no slowing.

TREATMENT

In the treatment of ventricular tachycardia the early recognition of the arrhythmia is most important, as this should be regarded as a medical emergency requiring prompt treatment. The choice of drug is generally between quinidine and procaine amide. As a rule procaine amide intravenously is used first, and the following procedures are carried out: An ampule containing 1 Gm. procaine amide is diluted to approximately 125 cc. with 5% dextrose in water. A vasopressor such as levarterenol is also available in another solution for intravenous use if any hypotension develops. The separate solutions of procaine amide and levarterenol are connected to the intravenous tubing so that either solution can be stopped or given when necessary. On the opposite arm a blood pressure cuff is attached, so that frequent blood pressure determinations may be made during the infusion. A direct-writing electrocardiograph, to permit constant monitoring of the electrocardiogram, is also used. Procaine amide is then started, and 1 Gm. is given over a period of approximately 30 minutes. If hypotension develops the procaine amide is stopped temporarily and replaced by the infusion of levarterenol (or other vasopressor).

At the first sign of reversion of the ventricular tachycardia to normal rhythm, procaine amide is discontinued. Occasionally the entire amount may be given without reversion to normal sinus rhythm. If such is the case, before giving an additional amount of procaine amide, it is preferable to wait one-half to one hour, or even longer, as reversion may take place during this period of waiting. If, after this period, however, the ventricular tachycardia persists, an additional gram of procaine amide may be given, diluted in the same manner as described, and with similar precautions. As a rule, the reversion takes place during infusion of the first gram of procaine amide. Once the rhythm has been reverted, I prefer oral quinidine for maintenance, generally giving 0.3 Gm. at intervals of approximately 6 hours.

The patient who initially presents with ventricular tachycardia and shock, or significant hypotension, should first be given a vasopressor agent, as we have seen some instances in which reversion has taken place merely by elevating the blood pressure, thereby making administration of procaine amide or other drugs unnecessary. In fact, any type of arrhythmia associated with shock or severe hypotension may possibly be reverted or aided by the initial use of a vasopressor agent. If ventricular tachycardia is a manifestation of digitalis toxicity, the use of potassium is indicated, with or without additional procaine amide. Quinidine is also effective in the treatment of ventricular tachycardia, using the same procedures and precautions as with procaine amide administration. When giving quinidine intravenously, 0.6 Gm. is diluted in 100–150 cc. of 5% dextrose in water, and the intravenous solution is given slowly over a period of approximately one-half hour. Reversion may take place after a small amount of the drug has been given; at the first sign of reversion the drug is discontinued. Occasionally larger amounts of quinidine are necessary, but one should allow a suitable lapse of time before starting with additional medication by the intravenous route. Procaine amide or quinidine may be administered intramuscularly, if desired, but the amount of the drug needed can be regulated more satisfactorily if the intravenous route is used. It must be appreciated that the intravenous administration of procaine amide, quinidine or

other drug that might be effective in eliminating ventricular tachycardia may be dangerous and may result in sudden death. The physician, therefore, must weigh the indications carefully before instituting this type of treatment. Whenever possible in the treatment of ventricular tachycardia, we prefer to use procaine amide intravenously; however, when oral medication is desired for treatment of the tachycardia, or maintenance, quinidine has appeared more effective.

Another medication that can control ventricular tachycardia is magnesium sulfate injected intravenously. This should also be given slowly as it entails some risk. The dosage will vary from 1–4 Gm., and reversion may take place within a matter of minutes.

Once reversion has taken place, the use of quinidine in doses of 0.3 Gm. orally every 4–6 hours is generally effective in the initial phase of maintenance. For long-range prophylaxis, 0.3 Gm. quinidine at approximately 6-hour intervals has proved satisfactory with most patients. The use of 0.2 Gm. 3 times a day (in our experience) is generally not as effective in preventing attacks of ventricular tachycardia as the 0.3 Gm. given at least 4 times a day and, in some cases, 5 or 6 times daily. In some resistant cases higher doses of quinidine may be necessary; 0.4–0.6 Gm. at intervals of 4–6 hours may be required. At times there is recurrence of tachycardia only during the night; if so, this indicates that the blood level of quinidine is ineffective at that time. The use of a theoretically longer-acting preparation, such as quinidine gluconate 0.3–0.6 Gm. at bedtime, can be tried; if this does not solve the problem, the patients are then advised to set an alarm clock to wake them up at a specified time during the night to take an additional dosage of quinidine. Procaine amide in doses of 250–500 mg. 3–6 times daily has also been effective, but it has been our clinical impression that oral quinidine has been superior as a prophylaxis to procaine amide or other drugs.

There are occasional cases of ventricular tachycardia that do not respond to any of the aforementioned treatments, and in such cases use of external electric countershock under general anesthesia has recently been reported successful in several patients (250–450 volts).

VENTRICULAR FIBRILLATION

This generally fatal arrhythmia is one in which there is no actual contraction of the ventricles but merely wormlike movements that occur after the heart stops contracting. Circulation ceases and death follows shortly thereafter. Naturally no heart sounds are produced during ventricular fibrillation, and the electrocardiogram reveals no myocardial contraction. Instead coarse chaotic waves consistent with ventricular fibrillation occur. The importance of closed chest cardiac massage recently developed at Johns Hopkins University is worthy of re-emphasis. Utilizing the method advocated by this group, external cardiac massage should be carried out with the patient supine, lying on a hard surface. The heel of one hand, with the other hand on top of it, is placed over the sternum, just above the xiphoid. Firm pressure is applied directly downward at about 60 times a minute. At the end of each pressure stroke the hands are lifted somewhat to allow full expansion of the chest. Mouth-to-mouth respiration should be carried out, if no other available means are present for maintaining an adequate airway; if two persons are present the massage and respiration can be carried out simultaneously. After this preliminary emergency treatment, defibrillation of the heart can then be attempted when the necessary equipment is available. Quick thinking on the part of the physician in such an emergency has been life-saving.

CONCLUDING REMARKS

By paying strict attention to all of the various aspects of the clinical evaluation of tachycardias, namely that of rate, first heart sound, rhythm and effect of carotid pressure, one may obtain valuable clues leading to the proper diagnosis and treatment of tachycardias. To illustrate: a patient may have paroxysmal tachycardia with a ventricular rate of 190; the rhythm is regular and the first sound does not change in intensity. The rate of 190 is generally too fast for that of normal sinus tachycardia, unless the patient is an infant or child or has high fever or thyrotoxicosis. If this were untreated atrial flutter, the usual patient would have a

2:1 A.V. block, making the atrial rate of 380. Since the flutter rate of the atria generally ranges between 250 and 350, an atrial rate of 380 is unusually fast, and therefore it is unlikely that the tachycardia in question is flutter with 2:1 A.V. block. High fever or thyrotoxicosis represents exceptions and may be associated with atrial flutter of more rapid rate. One patient with severe thyrotoxicosis had an atrial rate that exceeded 400, and in fact it was one of the first clues that one might be dealing with masked thyrotoxicosis in this otherwise apathetic patient.

Atrial fibrillation must be eliminated because the rhythm is noted to be perfectly regular, and the first heart sound did not change in intensity. A rate of 190, regular with a constant first heart sound, would be perfectly consistent with paroxysmal atrial tachycardia. Although the rate is consistent with ventricular tachycardia, slight irregularities of rhythm, plus change in intensity of the first heart sound, may frequently be present. This would tend to make this rhythm unlikely, but one could be further reassured by using carotid sinus pressure in the manner as described under atrial tachycardia. If it were paroxysmal atrial tachycardia, carotid sinus pressure might terminate the attack, thereby establishing the diagnosis. On the other hand, paroxysmal ventricular tachycardia would be completely unaffected by carotid sinus stimulation. If any slowing whatsoever was produced on carotid sinus stimulation, this would eliminate the most serious of the tachycardias, ventricular tachycardia. These simple deductions about the person having paroxysmal rapid heart action have proved most useful in suggesting or establishing the diagnosis, or narrowing the possibilities down to one or two. This method is particularly useful when electrocardiographic documentation is not available, but it is obvious that the electrocardiogram should always be used as the final analysis in diagnosis of tachycardia whenever possible. The electrocardiogram should also be recorded whenever carotid sinus pressure is being used, as this may be the only means of identifying the true underlying arrhythmia.

Appreciation is expressed to Miss Jonnie Morrow, secretary, and Mr. Bernard Salb, photographer, both of Georgetown University Hospital, for their valuable assistance in preparation of this manuscript.

REFERENCES

1. Levine, S. A., and Harvey, W. P.: *Clinical Auscultation of the Heart* (2d ed.; Philadelphia: W. B. Saunders Company, 1959), p. 98.
2. Harvey, W. P., and Levine, S. A.: The changing intensity of the first sound in auricular flutter: An aid to diagnosis by auscultation, Am. Heart J. 35:924-939, 1948.
3. Friedberg, C. K.: *Diseases of the Heart* (2d ed.; Philadelphia: W. B. Saunders Company, 1956), p. 344.
4. Lown, B., and Levine, S. A.: The carotid sinus: Clinical value of its stimulation, Circulation 23:766-789, 1961.
5. Wood, P.: *Diseases of the Heart and Circulation* (Philadelphia: J. B. Lippincott Company, 1956), p. 211.
6. Millikan, C. H.: Lewis A. Conner Lecture, 34th Scientific Session of the American Heart Association, Miami Beach, October, 1961.
7. Blumgart, H. L., *et al.*: What's new in arrhythmias?: A panel discussion, Circulation 20:286-297, 1959.
8. Kouwenhoven, S. B., Jude, J. R., and Knickerbocker, C. G.: Closed chest cardiac massage, J.A.M.A. 173:1064-1067, July 9, 1960.
9. Zoll, P. M., Linenthal, A. J., and Phelps, M. D., Jr.: Termination of refractory tachycardia by external electric countershock, 34th Scientific Session of the American Heart Association, Miami Beach, October, 1961.

INDEX

(*January-December 1961*)

F

G

Q

R